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POLYCYSTIC OVARY SYNDROME AND BONE TURNOVER IN POSTMENOPAUSAL WOMEN*

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Polycystic ovary syndrome (PCOS) is a common heterogeneous disease, whose pathogenesis is a complex set of mechanisms, including epigenetic factors and neuroendocrine dysfunction [1]. Considering the metabolic and hormonal changes associated with PCOS, its potential effect on bone health has been proposed [2]. However, this has a multidirectional effect, because hyperandrogenemia, central obesity and insulin resistance (IR) contribute to the preservation of bone mineral density (BMD), thereby protecting against the development of osteoporosis, while hormonal imbalance, chronic inflammation, hypovitaminosis D, increased cortisol levels, low estrogen levels contribute to its reduction [3]. While higher androgens levels, hyperinsulinemia and obesity, which are common in patients with PCOS, contributes to increased bone mineral density (BMD); statistical data does not support that this results in decreased risk of fractures [4]. As a result, inconsistent evidence exists as to whether PCOS is a bone-protecting or bone-deteriorating disorder [5].

The aim of the current study was to evaluate the effect of several hormonal and metabolic alterations associated with polycystic ovary syndrome on bone in postmenopausal women.

MATERIALS AND METHODS

This is a prospective study conducted from February 2021 to April 2022 at the Medical University Hospital, involving a total of 98 women aged 50–65 years. The main group consisted of 56 women with PCOS phenotype «A» (classic: hyperandrogenism, ovulatory dysfunction and polycystic ovarian morphology) who had complete information on the age and PCOS diagnosis (Rotterdam, 2003) [6] as well as control group (n = 42) age-matched women without this disease. Data on age, lifestyle, health status, and family history of chronic disease were collected from participants at the study. Insufficient physical activity was defined as physical activity less than two times per week. Exclusion criteria: history of fractures; osteoporosis treatment; other endocrine, hepatorenal disorders; long-term use of glucocorticoids and

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use of hormonal contraception or insulin sensitizing agents (metformin, thiazolidinediones, and myoinositol) for at least 3 months before enrollment; bad habits (alcohol abuse, excessive coffee consumption, tobacco smoking).

In all patients, bone mineral density, bone mineral metabolism and levels of the main calcium-regulating hormones, markers of osteoresorption and bone formation were studied. Dual-energy X-ray absorptiometry (DEXA, Hologic Discovery — QDR 4500A) was performed to determine BMD of the lumbar spine (L1–L4) and femoral neck. The interpretation of the obtained data was carried out according to WHO criteria using the T-score, where osteoporosis was defined as a T-score BMD range less than −2.0 SD (Standard Deviation); between −1.0 and −2.0 SD — osteopenia, and range between −1.0 and +2.5 SD were accepted as normal [7]. Serum levels of procollagen I C-terminal propeptide (P1NP) as a marker of bone formation and collagen type I C-terminal telopeptide (b-CTx) as a bone resorption marker were assessed using an enzyme-linked immunosorbent assay (ELISA, Roche, Switzerland). The levels of ionized calcium (Ca^{2+}) and phosphorus (P^{+}), parathyroid hormone (PTH), 25(OH) vitamin D ($25(OH)D_3$), anti-Mullerian hormone (AMH), insulin, testosterone (T), 17-hydroxyprogesterone (17-OHP), dehydro-epiandrostenedione sulfate (DHEAS), follicle stimulating hormone, luteinizing hormone and estradiol were determined. Homeostatic Model Assessment of insulin resistance (HOMA-IR) was used for quantifying insulin sensitivity and resistance, body mass index (BMI), waist circumference (WC), hip circumference (HC) and waist-to-hip ratio (WHR) were used to determine overweight and obesity [3].

The study protocol was approved by the Biomedical Ethics Committee at the Azerbaijan Medical University (No. 02/14 dated 10/14/2021), informed consent was obtained from all patients. The study was conducted in accordance with the World Medical Association Declaration of Helsinki.

Statistical data were analyzed by nonparametric testing using BioStat Pro 6.2.2.0 software (AnalystSoft Inc., Walnut, USA). Quantitative data are presented as median and quartiles 1 and 3 (Me [Q1; Q3]). The significance of intergroup differences was established using the Mann–Whitney U test ($p < 0.05$). To identify relationships between indicators, a univariate analysis was carried out with the calculation of the Spearman rank correlation coefficient (r) [8].

RESULTS AND THEIR DISCUSSION

According to the data presented in Table 1, noteworthy is the later age of menopause in women with PCOS ($p = 0.007$). Age of menopause was found to be 51.4 (45–58) years in PCOS and 49.7 (45–55) age in control subjects. Women with PCOS had a higher BMI than controls ($p = 0.009$); the mean BMI of 30.8 ± 2.6 kg/m^2 and the mean waist circumference of 104.3 ± 6.1 cm, demonstrate that PCOS group had a tendency toward overweight and obesity. Nutrients intake, sedentary time were associated with high WHR ($p < 0.05$), but sleep duration did not contribute to WHR. The prevalence of obesity by WHR > 0.85 was 73.2%.

Determination of $25(OH)D_3$ demonstrated a significant decrease in its level in patients with PCOS ($p = 0.036$), while in both groups it corresponded to the level of «insufficiency» with a reference interval of 30–100 ng/mL [9].

Also, in PCOS there was a tendency towards increased serum AMH levels ($p = 0.004$). Mean serum PTH levels were higher in patients with PCOS ($p = 0.041$), remaining within the reference range. In patients with PCOS, results show higher than normal levels of 17-OHP ($p = 0.025$), however, results are inside the reference interval. In patients with PCOS, serum testosterone levels were within the reference range and higher than in the control group ($p = 0.034$), in addition, there was a significant increase in DHEAS compared to the control group ($p = 0.042$).

Overall, the PCOS group had higher fasting insulin levels and HOMA-IR index than the control group. When analyzing the above-described biochemical markers in patients with PCOS, a positive correlation was established between testosterone levels, on the one hand, and insulin ($r = 0.329$; $p = 0.047$), HOMA-IR

(r = 0.325, p = 0.04) and PTH (r = 0.201, p = 0.031), on the other. It was revealed that the HOMA-IR index correlated with BMI (r = 0.319, p=0.04) and AMH levels (r = 0.527, p = 0.001).

Features of bone remodeling identified in the group of patients with PCOS were characterized by a slightly reduced or low-normal level of ionized calcium in the blood against the background of reference values of phosphatemia. At the same time, hypocalcemia was observed in 21.4% of PCOS patients, while in the control group this figure was 2 times less. Cases of hypocalcemia in the first group were predominantly moderate (p=0.026).

Analysis of the formation of markers and bone tissue resorption, in particular P1NP and b-CTx, as control components of the remodeling process, revealed significant deviations in observations from the control (p = 0.028 and

Table 1

Clinical and laboratory characteristics of the examined patients (Me [Q1; Q3])

Parameter	PCOS (n = 56)	Control (n = 42)	P
Age, year	54.4 [47.1;60.5]	55.6 [46.2;62.4]	ns
BMI, kg/m^2	31.2 [26.4;33.6]	27.8 [23.6;28.9]	0.009
Menopause, year	3.4 [1.6; 10.7]	5.1 [2.0;12.3]	0.007
Ca^{2+}, mmol/L	1.08 [1.01;1.14]	1.1 [1.09;1.18]	0.026
P^{+}, mmol/L	4.1 [3.3;4.8]	3.9 [2.8;4.2]	ns
Glucose, mmol/L	5.3 [4.7;5.6]	4.7 [4.2;5.1]	ns
Insulin, mIU/mL	12.4 [7.8;20.2]	7.9 [6.5;15.5]	0.003
HOMA-IR, U	2.9[2.4;7.1]	1.9 [1.3;3.6]	0.03
FSH, mIU/mL	72.8 [43.09;89.60]	77.1 [41.10;93.81]	0.036
LH, mIU/mL	39.2 [28.58;54.08]	22.0 [16.27;43.71]	0.029
AMH, ng/mL	0.364 [0.132;1.418]	0.11 [0.07;0.264]	0.004
Testosterone, ng/mL	0.57 [0.47;0.69]	0.42 [0.25;0.54]	0.034
17-OHP, ng/mL	0.5 [0.42;0.58]	0.33 [0.15;0.51]	0.025
DHEAS, ng/mL	1.36 [0.30;2.84]	1.0 [0.45;2.04]	0.042
25(OH)D_3, ng/mL	15.34 [8.01;32.86]	26.52 [11.84;37.50]	0.036
PTH, pg/mL	52.33 [41.3;61.9]	48.15 [40.2;59.5]	0.041
P1NP, ng/mL	41.17 [23.5;66.2]	47.70 [29.7;70.3]	0.028
b-CTx, ng/mL	5.38 [4.01;8.07]	5.11 [4.55;6.32]	0.032
DEXA (lumbar spine), SD	–1.63 [–2.2;1.1]	–1.98 [–2.4;0.05]	0.047
DEXA (femur neck), SD	–1.18 [–2.0;1.6]	–1.22 [–2.1;0.9]	ns

Notes:
AMH — anti-Mullerian hormone;
b-CTx — collagen type I C-terminal telopeptide;
BMI — body mass index;
DHEAS — dehydro-epiandrostenedione sulfate;
DEXA — dual-energy X-ray absorptiometry;
FSH — follicle stimulating hormone;
HOMA-IR — Homeostatic Model Assessment of insulin resistance;
LH — luteinizing hormone;
17-OHP — 17-hydroxyprogesterone;
25(OH)D_3 — vitamin D;
P1NP — procollagen I C-terminal propeptide;
PCOS — polycystic ovary syndrome;
PTH — parathyroid hormone;
SD — Standard Deviation.

p = 0.032, respectively). Positive relationship was found between BMI and the activity of the resorption marker b-CTx (r = 0.342, p = 0.002). PTH content correlated with P1NP and b-CTx markers with a coefficient r = –0.532, p = 0.001 and r = 0.413, p = 0.002 respectively. Also, in the group of patients with PCOS, a collinear relationship between the values of AMH and b-CTx was determined (r = –0.507, p = 0.001).

An osteodensitometric study revealed the prevalence of osteopenic syndrome in a group of patients with PCOS. Among women with PCOS, abnormal values of T-criteria were detected in 53.6%, while in the control group of a comparative level, BMD was detected in 52.4% of those examined. It should be noted that no differences were found in the distribution of densitometric parameters: in the main and control groups, in more than 50% of cases, the T-score at lumbar spine range for osteopenia.

Women with PCOS had higher T-score BMD values than the control group, however, statistically they differed only in the spine (p = 0.047). An inverse relationship has been established between the T-criterion in the L1–L4 region and the b-CTx level (r = –0.387, p = 0.026). In the PCOS group, a positive correlation was observed between lumbar spine BMD and insulin levels (r = 0.267, p = 0.042) and with AMH (r = 0.561, p = 0.031). There is a moderate correlation between BMD in the lumbar spine area and testosterone levels (r = 0.458, p = 0.039). BMD in women with PCOS was higher compared to the control group. This effect remained significant when excluding the two patients with low AMH values, but was limited to the spinal region.

Higher DHEAS levels were associated with higher femoral neck BMD (r = 0.473, p = 0.035). There was no statistically significant correlation between femoral neck BMD with total testosterone (r = 0.433; p > 0.5) and 17-OHP (r = 0.312; p > 0.1). Thus, neither total testosterone nor 17-OHP levels correlated with mineral density measurements, and the bone formation marker P1NP showed a negative correlation with femoral neck BMD (r = –0.429, p = 0.038).

The present study found that women with PCOS experienced menopause 2 to 3 years later than age-matched controls, which is consistent with several other studies [10]. Women with PCOS had lower FSH levels and higher AMH levels compared to controls. The exact mechanism responsible for high AMH levels in PCOS is not completely clear, but it is known that obesity, insulin resistance and hyperandrogenism play an important role in increasing AMH levels [11]. Also, AMH is associated with rapid bone loss [12]. The observed $25(OH)D_3$ deficiency may have a positive effect on the pathogenesis of PCOS and be an independent prognostic parameter for the condition of bone tissue in this syndrome [2]. Data on pronounced hypovitaminosis D with an increase in PTH levels in patients with PCOS are of particular interest, associated with the nature of the interaction of system regulators, in their role in ensuring calcium homeostasis and bone mineralization [9]. Additionally, postmenopausal women with PCOS had higher levels of DHEAS, total T, and 17-OHP than control women. This is confirmed evidence that the hyperandrogenism observed in women with PCOS persists after the transition to menopause [1].

Currently, we have identified significantly higher BMD values in the lumbar region in the group of women with PCOS compared to age-matched control women. A connection has been established between testosterone and lumbar spine BMD, and DHEAS with the BMD of the femoral neck, which corresponds to the data of some authors [13]. Taken together, these studies suggest possible anabolic effects of androgens on bone metabolism. Elevated androgen levels can cause insulin resistance and cause a number of metabolic disorders [3]. There is evidence that insulin can stimulate osteoblast differentiation, but elevated insulin levels in women with PCOS lead to insulin resistance, which contributes to decreased BMD [14]. This gives grounds to assert that obesity and hyperandrogenemia are factors that positively affect BMD, and is inconsistent with the results of a number of studies [15].

Data analysis showed a slightly higher activity of reparative osteogenesis in postmenopausal patients with PCOS compared to the comparison group. At the same time, in patients with PCOS, a less pronounced change in BMD was determined against the background of activation of bone remodeling processes.

These changes may contribute to the deterioration of bone microarchitecture, exacerbating the manifestations of menopausal osteoporosis. PCOS is thought to exacerbate changes in bone turnover, thereby promoting postmenopausal bone breakdown [4]. The results of studies by several authors have also shown that women with PCOS are at higher risk of fractures compared to the general population, despite normal or increased BMD, which suggests the existence of a number of PCOS-specific aberrations that degrade bone quality and may be important factors contributing to the development of osteoporosis [5]. The identified associations suggest that insulin resistance and hyperinsulinemia in women with PCOS may be factors that protect against the relative loss of bone mineral density, and therefore DEXA in this category of patients will be of little information, which also coincides with the opinion of a number of authors [10].

Thus, hyperandrogenism persists in women with PCOS for at least 10 years after menopause. Continued exposure to excess androgens in women with PCOS, which can lead to a variety of adverse clinical consequences. Our results highlight the importance of more detailed analyzes covering a broader range of current studies, which will require appropriate sample design and calculations to adequately address this issue.

CONCLUSION

Bone mineral density in women with polycystic ovary syndrome declines more slowly with age compared to their peers, possibly due to the effects of excess androgens and insulin. In postmenopausal women with polycystic ovary syndrome, metabolic processes in bone tissue accelerate, while bone resorption exceeds bone formation. Thus, women affected by polycystic ovary syndrome may be considered at risk of developing osteopenic syndrome. Although the diagnosis of osteoporosis is not based on laboratory tests, which including analysis of bone turnover markers, it seems advisable to include such testing in the examination algorithm for patients with polycystic ovary syndrome to diagnose metabolic changes in bone tissue that may contribute to the development of osteoporosis.

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Considering the metabolic and hormonal changes associated with polycystic ovary syndrome (PCOS), their potential affect bone health has been proposed. The **aim** of the current study was to evaluate the effect of several hormonal and metabolic alterations associated with polycystic ovary syndrome on bone in postmenopausal women.

Materials and methods: The study included 56 patients with PCOS and 42 women without this disease aged 50–65 years. Dual-energy X-ray absorptiometry was used to measure bone mineral density (BMD) of the lumbar spine and femoral neck. Serum levels of amino-terminal propeptide of procollagen 1 (P1NP) and carboxy-terminal telopeptide of type I collagen (b-CTx), as well as levels of parathyroid hormone (PTH), vitamin D (25(OH)D3), anti-Mullerian hormone (AMH), insulin, testosterone (T), 17-hydroxyprogesterone (17-OHP), dehydroepiandrostenedione sulfate (DHEAS), follicle stimulating hormone, luteinizing hormone, and estradiol were determined. The HOMA-IR index and anthropometric indicators were calculated.

Statistical data were analyzed by nonparametric testing using BioStat Pro 6.2.2.0 software (AnalystSoft Inc., Walnut, USA). Quantitative data are presented as median and quartiles 1 and 3 (Me [Q1; Q3]); The significance of intergroup differences was established using the Mann–Whitney U test ($p < 0.05$). To identify relationships between indicators, a univariate analysis was carried out with the calculation of the Spearman rank correlation coefficient (r).

Results. Women with PCOS had late-onset menopause ($p = 0.007$) and a higher body mass index (BMI, $p = 0.009$) than the control group. Additionally, women with PCOS had higher levels of DHEAS, total T, and 17-OHP than controls. P1NP and b-CTx were significantly different from controls ($p = 0.028$ and $p = 0.032$, respectively). A direct relationship was found between BMI and the activity of the resorption marker b-CTx ($r = 0.342$, $p = 0.002$) and an inverse collinear relationship between AMH values with b-CTx ($r = -0.507$, $p = 0.001$). BMD in PCOS was higher compared to controls. An inverse relationship exists between BMD of L1–L4 and b-CTx level ($r = -0.387$, $p = 0.026$) and positive with insulin ($r = 0.267$, $p = 0.042$), AMH ($r = 0.561$, $p = 0.031$) and testosterone ($r = 0.458$, $p = 0.039$). Higher DHEAS levels were associated with higher femoral neck BMD ($r = 0.473$, $p = 0.035$). P1NP was negatively correlated with femoral neck BMD ($r = -0.429$, $p = 0.038$).

Conclusions. Bone mineral density in women with polycystic ovary syndrome declines more slowly with age compared to their peers, possibly due to the effects of excess androgens and insulin. In postmenopausal women with polycystic ovary syndrome, metabolic processes in bone tissue accelerate, while bone resorption exceeds bone formation. Thus, women affected by polycystic ovary syndrome may be considered at risk of developing osteopenic syndrome.

Keywords: polycystic ovary syndrome; bone mineral density; markers of bone turnover.

СИНДРОМ ПОЛІКІСТОЗНИХ ЯЄЧНИКІВ І КІСТКОВИЙ ОБМІН У ЖІНОК У ПОСТМЕНОПАУЗІ

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Зважаючи на метаболічні та гормональні перебудови, пов'язані із синдромом полікістозних яєчників (СПКЯ), виникає припущення про їх потенційний вплив на здоров'я кісток. **Метою** даного дослідження було оцінити вплив певних гормональних та метаболічних змін, пов'язаних із синдромом полікістозних яєчників, на кістки у жінок у період постменопаузи.

Матеріали та методи. До дослідження було включено 56 пацієнток із СПКЯ та 42 жінки без даного захворювання у віці 50–65 років. Двоенергетичну рентгенівську абсорбціометрію використовували для вимірювання мінеральної щільності кістки (МЩК) поперекового відділу хребта та шийки стегна. За допомогою імуноферментних методів у сироватці крові визначали: аміно-кінцевий пропептид проколагену 1 (P1NP) і карбокси-кінцевий телопептид колагену типу I (b-CTx), а також паратиреоїдний гормон (ПТГ), вітамін D (25(OH)D_3), антимюллерів гормон (АМГ), інсулін, тестостерон (Т), 17-гідроксипрогестерон (17-ОНР), дегідроепіандростендіон-сульфат (DHEAS), лютеїнізуючий і фолікулостимулюючий гормони та естрадіол. Розраховували індекс HOMA-IR та антропометричні показники.

Статистичні дані аналізували за допомогою непараметричного тестування з використанням програмного забезпечення BioStat Pro 6.2.2.0 (AnalystSoft Inc., Walnut, США). Кількісні дані представлені у вигляді медіани та квартилів 1 і 3 (Me [Q1; Q3]); Достовірність міжгрупових відмінностей встановлювали за U-критерієм Манна–Уітні ($p < 0,05$). Для виявлення зв'язків між показниками проводили однофакторний аналіз з розрахуванням коефіцієнту рангової кореляції Спірмана (r).

Результати. У жінок із СПКЯ виявлено пізніший вік настання менопаузи ($p = 0,007$) та більш високий індекс маси тіла (IMT, $p = 0,009$), ніж у контрольній групі. Крім того, жінки із СПКЯ мали більш високі рівні DHEAS, загального Т та 17-ОНР, ніж група контролю. Рівні P1NP і b-CTx значно відрізнялися від контрольних ($p = 0,028$ і $p = 0,032$ відповідно). Було виявлено прямий зв'язок між IMT та активністю маркера резорбції b-CTx ($r = 0,342$, $p = 0,002$) та зворотну колінеарну залежність між значеннями АМГ та b-CTx ($r = -0,507$, $p = 0,001$). МЩК за СПКЯ, порівняно з контролем, була вищою. Встановлено зворотний зв'язок між МЩК в області L1–L4 та рівнем b-CTx ($r = -0,387$, $p = 0,026$) та позитивний — з інсуліном ($r = 0,267$, $p = 0,042$), АМГ ($r = 0,561$, $p = 0,031$) та тестостероном ($r = 0,458$, $p = 0,039$). Вищі рівні DHEAS пов'язані з вищою МЩК шийки стегнової кістки ($r = 0,473$, $p = 0,035$). P1NP показав негативну кореляцію з МЩК шийки стегнової кістки ($r = -0,429$, $p = 0,038$).

Висновки. Мінеральна щільність кісток у жінок із синдромом полікістозних яєчників знижується з віком повільніше порівняно з їхніми однолітками, можливо, через вплив надлишку андрогенів та інсуліну. Однак у постменопаузальному періоді у жінок із синдромом полікістозних яєчників обмінні процеси в кістковій тканині прискорюються, при цьому резорбція кістки перевищує кісткоутворення. Таким чином, пацієнти з синдромом полікістозних яєчників можуть розглядатися як група ризику розвитку остеопенічного синдрому.

Ключові слова: синдром полікістозних яєчників; мінеральна щільність кістки; маркери кісткового обміну.